L3	('p-selectin' or cd62 or psgl$)same hypertension	32	L3
L2	('p-selectin' or cd62 or psgl$) and hypertension	402	L2
L1	eppihimer.in.	6	L1

END OF SEARCH HISTORY

```
 begin 5,73,155,399
       28aug04 10:12:13 User208760 Session D2507.2
            $0.00    0.078 DialUnits File410
     $0.00  Estimated cost File410
     $0.02  TELNET
     $0.02  Estimated cost this search
     $0.31  Estimated total session cost   0.161 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R) 1969-2004/Aug W4
         (c) 2004 BIOSIS
  File  73:EMBASE 1974-2004/Aug W4
         (c) 2004 Elsevier Science B.V.
  File 155:MEDLINE(R) 1951-2004/Aug W4
         (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.
  File 399:CA SEARCH(R) 1967-2004/UD=14109
         (c) 2004 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
Alert feature enhanced for multiple files, etc. See HELP ALERT.

      Set  Items  Description
      ---  -----  -----------
? e au=eppihimer

Ref    Items  Index-term
E1         1  AU=EPPIGER E N
E2         1  AU=EPPIGER, E. N.
E3         0 *AU=EPPIHIMER
E4         3  AU=EPPIHIMER L A
E5         2  AU=EPPIHIMER L.A.
E6         1  AU=EPPIHIMER LOIS A
E7         2  AU=EPPIHIMER LOIS ANN
E8         7  AU=EPPIHIMER M
E9        29  AU=EPPIHIMER M J
E10        2  AU=EPPIHIMER M.
E11       17  AU=EPPIHIMER M.J.
E12        3  AU=EPPIHIMER MICHAEL

          Enter P or PAGE for more
? p

Ref    Items  Index-term
E13       21  AU=EPPIHIMER MICHAEL J
E14        1  AU=EPPIHIMER, LOIS A.
E15        2  AU=EPPIHIMER, LOIS ANN
E16        1  AU=EPPIHIMER, M.
E17        1  AU=EPPIHIMER, MICHAEL
E18       17  AU=EPPIHIMER, MICHAEL J.
E19        1  AU=EPPINER E A
E20        1  AU=EPPINETTE J
E21        1  AU=EPPINETTE R T
E22        1  AU=EPPINETTE W
E23        2  AU=EPPING A
E24        3  AU=EPPING A.

          Enter P or PAGE for more
? s e8-e18
                7  AU=EPPIHIMER M
               29  AU=EPPIHIMER M J
                2  AU=EPPIHIMER M.
               17  AU=EPPIHIMER M.J.
                3  AU=EPPIHIMER MICHAEL
```

```
              21  AU=EPPIHIMER MICHAEL J
               1  AU=EPPIHIMER, LOIS A.
               2  AU=EPPIHIMER, LOIS ANN
               1  AU=EPPIHIMER, M.
               1  AU=EPPIHIMER, MICHAEL
              17  AU=EPPIHIMER, MICHAEL J.
      S1     101  E8-E18
? s s1 and (p(w)selectin or psgl? or cd62)
             101  S1
         4616882  P
           34569  SELECTIN
           13485  P(W)SELECTIN
            1340  PSGL?
            1055  CD62
      S2       39  S1 AND (P(W)SELECTIN OR PSGL? OR CD62)
? rd s2
...completed examining records
      S3       19  RD S2 (unique items)
? s s3 and hypertension
              19  S3
          658557  HYPERTENSION
      S4        0  S3 AND HYPERTENSION
? s (p(w)selectin or psgl? or cd62) and hypertension
         4616882  P
           34569  SELECTIN
           13485  P(W)SELECTIN
            1340  PSGL?
            1055  CD62
          658557  HYPERTENSION
      S5      333  (P(W)SELECTIN OR PSGL? OR CD62) AND HYPERTENSION
? s (p(w)selectin or psgl? or cd62) (20n)hypertension
         4616882  P
           34569  SELECTIN
           13485  P(W)SELECTIN
            1340  PSGL?
            1055  CD62
          658557  HYPERTENSION
      S6      120  (P(W)SELECTIN OR PSGL? OR CD62) (20N)HYPERTENSION
? rd s6
...examined 50 records  (50)
...examined 50 records  (100)
...completed examining records
      S7       67  RD S6 (unique items)
? s (p(w)selectin or psgl? or cd62) (20n)hypertension(20n)(inhibit? or prevent? or
suppress? or treat? or therap?)
Processing
Processing
         4616882  P
           34569  SELECTIN
           13485  P(W)SELECTIN
            1340  PSGL?
            1055  CD62
          658557  HYPERTENSION
         4072675  INHIBIT?
         2154353  PREVENT?
          825361  SUPPRESS?
         6687018  TREAT?
         6292520  THERAP?
      S8       49  (P(W)SELECTIN OR PSGL? OR CD62)
                   (20N)HYPERTENSION(20N)(INHIBIT? OR PREVENT? OR SUPPRESS?
                   OR TREAT? OR THERAP?)
? rd s8
...completed examining records
      S9       29  RD S8 (unique items)
```

```
? s s9 and py<2001
Processing
Processing
             29  S9
       50592248  PY<2001
     S10       8  S9 AND PY<2001
? t s10/3/all

 10/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0012853147   BIOSIS NO.: 200100024986
Increased plasma P-selectin and decreased thrombomodulin in
  pulmonary arterial hypertension were improved by continuous
  prostacyclin therapy
AUTHOR: Sakamaki Fumio (Reprint); Kyotani Shingo; Nagaya Noritoshi; Sato
  Nagato; Oya Hideo; Satoh Toru; Nakanishi Norifumi
AUTHOR ADDRESS: Division of Cardiology and Pulmonary Circulation,
  Department of Medicine, National Cardiovascular Center, 5-7-1
  Fujishirodai, Suita-shi, Osaka, 565-8565, Japan**Japan
JOURNAL: Circulation  102 (22): p2720-2725 November 28, 2000 2000
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 10/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0012159686   BIOSIS NO.: 199900419346
Platelet hyperactivity in hypertensive older patients is controlled by
  lowering blood pressure
AUTHOR: Riondino Silvia; Pignatelli Pasquale; Pulcinelli Fabio M; Lenti
  Luisa; Di Veroli Claudio; Marigliano Vincenzo; Gazzaniga Pier Paolo
  (Reprint)
AUTHOR ADDRESS: Dipartimento di Medicina Sperimentale e Patologia,
  Universia degli Studi di Roma "La Sapienza", Viale Regina Elena 324,
  00161, Roma, Italy**Italy
JOURNAL: Journal of the American Geriatrics Society  47 (8): p943-947 Aug.,
1999 1999
MEDIUM: print
ISSN: 0002-8614
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 10/3/3     (Item 3 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0011785175   BIOSIS NO.: 199900044835
Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent
  platelet activation infants with persistent pulmonary hypertension of the
  newborn
AUTHOR: Christou Helen (Reprint); Magnani Barbarajean; Morse David S;
  Allred Elizabeth N; Van Marter Linda J; Wessel David L; Kourembanas
  Stella
AUTHOR ADDRESS: Dep. Pediatrics, Div. Newborn Med. Dev. Newborn Biol.,
  Children's Hosp., 300 Longwood Ave., Enders 9, Boston, MA 02115, USA**USA
```

JOURNAL: Pediatrics 102 (6): p1390-1393 Dec., 1998 ***1998***
MEDIUM: print
ISSN: 0031-4005
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07510872 EMBASE No: 1998410959
Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent platelet activation in infants with persistent pulmonary hypertension of the newborn
Christou H.; Magnani B.; Morse D.S.; Allred E.N.; Van Marter L.J.; Wessel D.L.; Kourembanas S.
Dr. S. Kourembanas, Department of Pediatrics, Division of Newborn Medicine, Children's Hospital, 300 Longwood Ave, Boston, MA 02115 United States
Pediatrics (PEDIATRICS) (United States) 1998, 102/6 (1390-1393)
CODEN: PEDIA ISSN: 0031-4005
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 25

10/3/5 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07062983 EMBASE No: 1997344846
Evidence of platelet activation in hypertension
Blann A.D.; Lip G.Y.H.; Islim I.F.; Beevers D.G.
Dr. A.D. Blann, Haemost Thromb Vascular Biology Unit, Department of Medicine, The City Hospital, Duduley Road, Birmingham B18 7QH United Kingdom
Journal of Human Hypertension (J. HUM. HYPERTENS.) (United Kingdom) 1997, 11/9 (607-609)
CODEN: JHHYE ISSN: 0950-9240
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 5

10/3/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10369368 PMID: 7533336
Amyloid beta-protein precursor-rich platelet microparticles in thrombotic disease.
Nomura S; Komiyama Y; Miyake T; Miyazaki Y; Kido H; Suzuki M; Kagawa H; Yanabu M; Takahashi H; Fukuhara S
First Department of Internal Medicine, Kansai Medical University, Osaka, Japan.
Thrombosis and haemostasis (GERMANY) Oct **1994**, 72 (4) p519-22, ISSN 0340-6245 Journal Code: 7608063
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

10/3/7 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130011019 CA: 130(2)11019s PATENT
Methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome using dehydroepiandrosterone-3-sulfate
INVENTOR(AUTHOR): Araneo, Barbara A.; Orlinska, Urszula; Farrukh, Imad S.
LOCATION: USA
ASSIGNEE: University of Utah Research Foundation; Pharmadigm Inc.
PATENT: United States ; US 5846963 A DATE: 19981208
APPLICATION: US 516540 (19950818) *US 480744 (19950607) *US 480745 (19950607) *US 480748 (19950607) *US 480747 (19950607)
PAGES: 23 pp., Cont.-in-part of U.S. 5,587,369. CODEN: USXXAM
LANGUAGE: English CLASS: 514178000; A61K-031/56A

10/3/8 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122102975 CA: 122(9)102975b JOURNAL
Modification of leukocyte adhesion in spontaneously hypertensive rats by adrenal corticosteroids
AUTHOR(S): Suzuki, Hidekazu; Zweifach, Benjamin W.; Forrest, Michael J.; Schmid-Schoenbein, Geert W.
LOCATION: Inst. Biomedical Eng., Univ. California, San Diego, La Jolla, CA, USA
JOURNAL: J. Leukocyte Biol. DATE: 1995 VOLUME: 57 NUMBER: 1 PAGES: 20-6 CODEN: JLBIE7 ISSN: 0741-5400 LANGUAGE: English
? t s10/7/all

10/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012853147 BIOSIS NO.: 200100024986
Increased plasma **P-selectin** and decreased thrombomodulin in pulmonary arterial **hypertension** were improved by continuous prostacyclin **therapy**
AUTHOR: Sakamaki Fumio (Reprint); Kyotani Shingo; Nagaya Noritoshi; Sato Nagato; Oya Hideo; Satoh Toru; Nakanishi Norifumi
AUTHOR ADDRESS: Division of Cardiology and Pulmonary Circulation, Department of Medicine, National Cardiovascular Center, 5-7-1 Fujishirodai, Suita-shi, Osaka, 565-8565, Japan**Japan
JOURNAL: Circulation 102 (22): p2720-2725 November 28, 2000 **2000**
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background-Thrombosis in situ related to endothelial cell injury may contribute to the development of pulmonary hypertension (PH). P-selectin, a leukocyte adhesion receptor present in endothelial cells and platelets, reflects endothelial injury and platelet activation, and thrombomodulin (TM), a receptor for thrombin and a major anticoagulant proteoglycan on the endothelial membrane, reflects the anticoagulant activity of the endothelium. Methods and Results-To assess abnormal coagulation due to endothelial injury in patients with PH, plasma levels of soluble P-selectin and TM were measured in 32 patients with primary PH (PPH), 25 with secondary pulmonary arterial hypertension (sPAH), 31 with

pulmonary venous hypertension (PVH), and 17 healthy subjects (Control). These measurements were repeated after continuous infusion of prostacyclin in 15 patients with PPH and 3 with sPAH. P-selectin levels in both the sPAH and PPH groups were significantly higher than those in the Control and PVH groups ($P<0.05$). Plasma TM level in the PPH group was significantly lower than those in the other groups ($P<0.01$). After prostacyclin therapy, the lower TM level was increased and the higher P-selectin level was decreased ($P<0.05$). Conclusions-Decreased TM and increased P-selectin in PPH and sPAH may reflect in situ thrombosis due to endothelial injury. Prostacyclin may act not only as a vasodilator but also as an agent that improves endothelial injury and altered hemostasis in pulmonary arterial injury.

10/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012159686 BIOSIS NO.: 199900419346
Platelet hyperactivity in hypertensive older patients is controlled by lowering blood pressure
AUTHOR: Riondino Silvia; Pignatelli Pasquale; Pulcinelli Fabio M; Lenti Luisa; Di Veroli Claudio; Marigliano Vincenzo; Gazzaniga Pier Paolo (Reprint)
AUTHOR ADDRESS: Dipartimento di Medicina Sperimentale e Patologia, Universia degli Studi di Roma "La Sapienza", Viale Regina Elena 324, 00161, Roma, Italy**Italy
JOURNAL: Journal of the American Geriatrics Society 47 (8): p943-947 Aug., 1999 **1999**
MEDIUM: print
ISSN: 0002-8614
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: OBJECTIVE: Patients with hypertension tend to have a high prevalence of atherothrombotic accidents. Platelet hyperactivity is frequently associated with hypertension. Because the vascular disease associated with hypertension evolves over the years, we investigated platelet activity parameters in a population of older hypertensive patients with no other risk factors for cardiovascular disease. PARTICIPANTS: We studied 34 older, nonsmoking patients (mean age 74 +- 5 years) with uncomplicated **hypertension** before and after the normalization of blood pressure (BP) was achieved with the angiotensin-converting enzyme **inhibitor** quinapril alone or in combination with the Ca2+ antagonist nifedipine. MEASUREMENTS: Platelet aggregation, **P-selectin** (**CD62**) expression on the platelet surface, serum levels of Interleukin-1beta (IL-1beta) and of Interleukin-6 (IL-6), as well as plasma levels of soluble P-selectin and Endothelin-1 (ET-1), were analyzed. RESULTS: All platelet hyperactivity parameters werereduced significantly with the normalization of BP at the end of antihypertensive drug treatment (systolic/diastolic: 186.2 +- 2.7/103.4 +- 1.1 mm Hg vs 135.0 +- 1.3/85.9 U; 1.9 mm Hg; P < 001). Those factors more strictly associated with endothelium injury, such as ET-1 and IL-6, did not show variations. A significant correlation (Spearman Rank test) was observed among all platelet function parameters and blood pressure values. CONCLUSIONS: This study demonstrated that even in a population of older hypertensive patients with no other risk factor for atherogenic disease, normalization of blood pressure induces a significant reduction of the parameters of enhanced platelet hyperactivity independent of the action exerted, at the platelet level, by the antihypertensive drugs.

10/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011785175 BIOSIS NO.: 199900044835
Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent platelet activation infants with persistent pulmonary hypertension of the newborn
AUTHOR: Christou Helen (Reprint); Magnani Barbarajean; Morse David S; Allred Elizabeth N; Van Marter Linda J; Wessel David L; Kourembanas Stella
AUTHOR ADDRESS: Dep. Pediatrics, Div. Newborn Med. Dev. Newborn Biol., Children's Hosp., 300 Longwood Ave., Enders 9, Boston, MA 02115, USA**USA
JOURNAL: Pediatrics 102 (6): p1390-1393 Dec., 1998 ***1998***
MEDIUM: print
ISSN: 0031-4005
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objective. To investigate the effect of inhaled nitric oxide (NO) **treatment** in newborns with persistent pulmonary hypertension on adenosine 5'-diphosphate (ADP)-dependent platelet activation. Methods. After parental informed consent, infants with persistent pulmonary **hypertension** of the newborn were randomly assigned to receive conventional **treatment** (control group) or **treatment** with 40 parts per million of inhaled NO. Platelet activation was measured at time of entry and 30 minutes later by surface expression of **P-selectin** in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20 muM) using fluorescence-activated flow cytometry. Results. We examined 11 infants in the inhaled NO group and 13 in the control group. P-selectin expression, quantified as mean fluorescence, was not significantly different in the two groups of patients at baseline. Median percent change from baseline fluorescence was assessed using the Wilcoxon matched-pairs signed-rank test. At 30 minutes after enrollment there were no statistically significant changes from baseline fluorescence in either group of patients and at all ADP concentrations. Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not **inhibit** ADP-dependent platelet activation as measured by surface expression of **P-selectin** in infants with persistent pulmonary ***hypertension*** of the newborn.

10/7/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07510872 EMBASE No: 1998410959
Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent platelet activation in infants with persistent pulmonary hypertension of the newborn
Christou H.; Magnani B.; Morse D.S.; Allred E.N.; Van Marter L.J.; Wessel D.L.; Kourembanas S.
Dr. S. Kourembanas, Department of Pediatrics, Division of Newborn Medicine, Children's Hospital, 300 Longwood Ave, Boston, MA 02115 United States
Pediatrics (PEDIATRICS) (United States) 1998, 102/6 (1390-1393)
CODEN: PEDIA ISSN: 0031-4005
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 25

Objective. To investigate the effect of inhaled nitric oxide (NO) **treatment** in newborns with persistent pulmonary hypertension on

adenosine 5'- diphosphate (ADP)-dependent platelet activation. Methods. After parental informed consent, infants with persistent pulmonary **hypertension** of the newborn were randomly assigned to receive conventional **treatment** (control group) or **treatment** with 40 parts per million of inhaled NO. Platelet activation was measured at time of entry and 30 minutes later by surface expression of **P-selectin** in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20 muM) using fluorescence-activated flow cytometry. Results. We examined 11 infants in the inhaled NO group and 13 in the control group. P-selectin expression, quantified as mean fluorescence, was not significantly different in the two groups of patients at baseline. Median percent change from baseline fluorescence was assessed using the Wilcoxon matched-pairs signed-rank test. At 30 minutes after enrollment there were no statistically significant changes from baseline fluorescence in either group of patients and at all ADP concentrations. Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not **inhibit** ADP-dependent platelet activation as measured by surface expression of **P-selectin** in infants with persistent pulmonary ***hypertension*** of the newborn.

10/7/5 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07062983 EMBASE No: 1997344846
Evidence of platelet activation in hypertension
Blann A.D.; Lip G.Y.H.; Islim I.F.; Beevers D.G.
Dr. A.D. Blann, Haemost Thromb Vascular Biology Unit, Department of Medicine, The City Hospital, Duduley Road, Birmingham B18 7QH United Kingdom
Journal of Human Hypertension (J. HUM. HYPERTENS.) (United Kingdom) 1997, 11/9 (607-609)
CODEN: JHHYE ISSN: 0950-9240
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 5

To test the hypothesis that platelet activation is present is **hypertension**, we measured plasma markers beta thromboglobulin and soluble **P-selectin** in hypertensive patients and normotensive controls. Both markers were raised in the patients ($P < 0.05$), and in a subgroup of patients, beta thromboglobulin was reduced with successful **treatment** of **hypertension** with the ACE **inhibitor** quinapril. We suggest that reversible platelet activation is present in hypertension. This may be a contributing factor to the link between this risk factor and the development of thrombotic disease such as stroke.

10/7/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10369368 PMID: 7533336
Amyloid beta-protein precursor-rich platelet microparticles in thrombotic disease.
Nomura S; Komiyama Y; Miyake T; Miyazaki Y; Kido H; Suzuki M; Kagawa H; Yanabu M; Takahashi H; Fukuhara S
First Department of Internal Medicine, Kansai Medical University, Osaka, Japan.
Thrombosis and haemostasis (GERMANY) Oct **1994**, 72 (4) p519-22, ISSN 0340-6245 Journal Code: 7608063
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
We investigated the association of amyloid beta-protein precursor (APP) and platelet derived microparticles in 20 normal controls and 91 patients with various diseases causing a thrombotic tendency. Compared with the controls, the mean percentage of APP-positive microparticles was significantly greater in the patients with cerebral infarction (39.1 +/- 17.7%, p < 0.001), diabetes (31.1 +/- 12.6%, p < 0.001), and uremia (30.1 +/- 14.7%, p < 0.01), but not in those with hypertension (8.2 +/- 6.3%, p = NS). Sixteen patients with cerebral infarction, 20 with diabetes, and 11 with uremia had microparticles with very high APP levels. In normal controls, 7.2 +/- 3.7% of the microparticles were positive for P-selectin, while the percentage in cerebral infarction, diabetes, uremia, and hypertension was respectively 43.5 +/- 15.1%, 40.0 +/- 12.8%, 31.8 +/- 12.2%, and 11.6 +/- 7.3%. There was a significant correlation between P-selectin and APP positivity of microparticles. Our results suggest that microparticle APP may have a regulatory influence on coagulation abnormalities.
Record Date Created: 19950331
Record Date Completed: 19950331

10/7/7 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130011019 CA: 130(2)11019s PATENT
Methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome using dehydroepiandrosterone-3-sulfate
INVENTOR(AUTHOR): Araneo, Barbara A.; Orlinska, Urszula; Farrukh, Imad S.
LOCATION: USA
ASSIGNEE: University of Utah Research Foundation; Pharmadigm Inc.
PATENT: United States ; US 5846963 A DATE: 19981208
APPLICATION: US 516540 (19950818) *US 480744 (19950607) *US 480745 (19950607) *US 480748 (19950607) *US 480747 (19950607)
PAGES: 23 pp., Cont.-in-part of U.S. 5,587,369. CODEN: USXXAM
LANGUAGE: English CLASS: 514178000; A61K-031/56A
SECTION:
CA202004 Mammalian Hormones
IDENTIFIERS: tissue necrosis prevention dehydroepiandrosterone sulfate, reperfusion injury prevention dehydroepiandrosterone sulfate, bacterial translocation prevention dehydroepiandrosterone sulfate, adult respiratory distress syndrome prevention dehydroepiandrosterone sulfate, ischemia effects prevention dehydroepiandrosterone sulfate
DESCRIPTORS:
Burn...
chemical and thermal; method for preventing or reducing loss of tissue viability following injury using dehydroepiandrosterone-3-sulfate
P-selectin...
expression by platelets and endothelial cells; methods for preventing or reducing adherence of blood cells and platelets to endothelial cells and pulmonary hypertension using dehydroepiandrosterone-3-
Neutrophil...
method for preventing or reducing loss of tissue viability caused by adhesion of neutrophils to endothelial cells using dehydroepiandrosterone-3-sulfate
Myocardial infarction... Surgery... Trauma... Wound...
method for preventing or reducing loss of tissue viability following injury using dehydroepiandrosterone-3-sulfate
Injury...
method for preventing or reducing the effects of ischemia associated with injury using dehydroepiandrosterone-3-sulfate
Anti-ischemic agents...

method for preventing or reducing the effects of ischemia using dehydroepiandrosterone-3-sulfate
Antihypertensives...
methods for preventing or reducing adherence of blood cells and platelets to endothelial cells and pulmonary hypertension using dehydroepiandrosterone-3-sulfate
Blood cells... Cell adhesion... Platelet adhesion... Pulmonary hypertension ... Vascular endothelium...
methods for preventing or reducing bacterial translocation, adult respiratory distress syndrome, adherence of blood cells and platelets to endothelial cells and pulmonary hypertension using dehydroepi
Adult respiratory distress syndrome... Antibacterial agents...
Bacteria(Eubacteria)... Necrosis... Reperfusion injury...
methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome using dehydroepiandrosterone-3-sulfate
Hemorrhagic shock...
methods for treating hemorrhagic shock using dehydroepiandrosterone-3-sulfate
CAS REGISTRY NUMBERS:
651-48-9 methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome using dehydroepiandrosterone-3-sulfate

10/7/8 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122102975 CA: 122(9)102975b JOURNAL
Modification of leukocyte adhesion in spontaneously hypertensive rats by adrenal corticosteroids
AUTHOR(S): Suzuki, Hidekazu; Zweifach, Benjamin W.; Forrest, Michael J.; Schmid-Schoenbein, Geert W.
LOCATION: Inst. Biomedical English, University California, San Diego, La Jolla, CA, USA
JOURNAL: J. Leukocyte Biol. DATE: 1995 VOLUME: 57 NUMBER: 1 PAGES: 20-6 CODEN: JLBIE7 ISSN: 0741-5400 LANGUAGE: English
SECTION:
CA214005 Mammalian Pathological Biochemistry
CA202XXX Mammalian Hormones
IDENTIFIERS: corticosteroid modification leukocyte adhesion spontaneous hypertension, glucocorticoid endothelial cell leukocyte interaction hypertension
DESCRIPTORS:
Neutrophil...
glucocorticoid modification of activated neutrophil count in spontaneous hypertension
Adhesion,bio-... Corticosteroids,gluco-,biological studies...
Hypertension,spontaneous...
glucocorticoid modification of leukocyte adhesion in spontaneous hypertension
Leukocyte... Receptors,P-selectins... Vein,venule, endothelium...
glucocorticoid suppression of P-selectin-mediated leukocyte-endothelial interaction in spontaneous hypertension
CAS REGISTRY NUMBERS:
51-45-6 biological studies, glucocorticoid modification of histamine-induced leukocyte-endothelial interaction in mesenteric venules in spontaneous hypertension
50-23-7 glucocorticoid modification of leukocyte adhesion in spontaneous hypertension
? t s10/kwic/all
>>>KWIC option is not available in file(s): 399

10/KWIC/1 (Item 1 from file: 5)
DIALOG(R)File 5:(c) 2004 BIOSIS. All rts. reserv.

Increased plasma **P-selectin** and decreased thrombomodulin in pulmonary arterial **hypertension** were improved by continuous prostacyclin **therapy**
2000

10/KWIC/2 (Item 2 from file: 5)
DIALOG(R)File 5:(c) 2004 BIOSIS. All rts. reserv.

1999

...ABSTRACT: disease. PARTICIPANTS: We studied 34 older, nonsmoking patients (mean age 74 +- 5 years) with uncomplicated **hypertension** before and after the normalization of blood pressure (BP) was achieved with the angiotensin-converting enzyme **inhibitor** quinapril alone or in combination with the Ca2+ antagonist nifedipine. MEASUREMENTS: Platelet aggregation, **P-selectin** (**CD62**) expression on the platelet surface, serum levels of Interleukin-1beta (IL-1beta) and of Interleukin...

10/KWIC/3 (Item 3 from file: 5)
DIALOG(R)File 5:(c) 2004 BIOSIS. All rts. reserv.

1998

ABSTRACT: Objective. To investigate the effect of inhaled nitric oxide (NO) **treatment** in newborns with persistent pulmonary hypertension on adenosine 5'-diphosphate (ADP)-dependent platelet activation. Methods. After parental informed consent, infants with persistent pulmonary **hypertension** of the newborn were randomly assigned to receive conventional **treatment** (control group) or **treatment** with 40 parts per million of inhaled NO. Platelet activation was measured at time of entry and 30 minutes later by surface expression of **P-selectin** in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20...

...ADP concentrations. Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not **inhibit** ADP-dependent platelet activation as measured by surface expression of **P-selectin** in infants with persistent pulmonary ***hypertension*** of the newborn.

10/KWIC/4 (Item 1 from file: 73)
DIALOG(R)File 73:(c) 2004 Elsevier Science B.V. All rts. reserv.

Objective. To investigate the effect of inhaled nitric oxide (NO) **treatment** in newborns with persistent pulmonary hypertension on adenosine 5'- diphosphate (ADP)-dependent platelet activation. Methods. After parental informed consent, infants with persistent pulmonary **hypertension** of the newborn were randomly assigned to receive conventional **treatment** (control group) or **treatment** with 40 parts per million of inhaled NO. Platelet activation was measured at time of entry and 30 minutes later by surface expression of **P-selectin** in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20...

...ADP concentrations. Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not **inhibit** ADP-dependent platelet activation as measured by surface expression of **P-selectin** in infants with persistent pulmonary ***hypertension*** of the newborn.

1998

10/KWIC/5 (Item 2 from file: 73)
DIALOG(R)File 73:(c) 2004 Elsevier Science B.V. All rts. reserv.

To test the hypothesis that platelet activation is present is **hypertension**, we measured plasma markers beta thromboglobulin and soluble **P-selectin** in hypertensive patients and normotensive controls. Both markers were raised in the patients ($P < 0.05$), and in a subgroup of patients, beta thromboglobulin was reduced with successful **treatment** of **hypertension** with the ACE **inhibitor** quinapril. We suggest that reversible platelet activation is present in hypertension. This may be a...
1997

10/KWIC/6 (Item 1 from file: 155)
DIALOG(R)File 155:(c) format only 2004 The Dialog Corp. All rts. reserv.

Oct **1994**,
; Diabetes Mellitus--complications--CO; Disease Susceptibility; Factor IXa--antagonists and **inhibitors**--AI; Factor Xa--antagonists and **inhibitors**--AI; **Hypertension**--blood--BL; **Hypertension** --complications--CO; **P-Selectin**; Platelet Membrane Glycoproteins--blood--BL; Thrombosis--etiology--ET; Uremia--complications --CO
?